

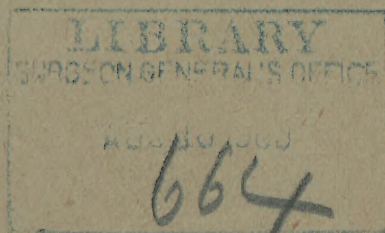
Corning (J. L.)

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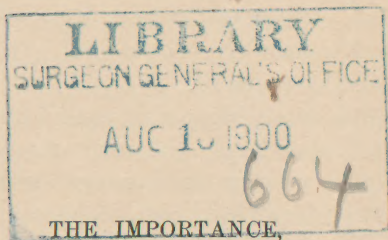
BY

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BOTH MEDICO-LEGAL AND CLINICAL, OF THE
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By J. LEONARD CORNING, A. M., M. D.

WHATEVER our hopes or our misgivings touching the curability of certain organic diseases of the nervous system, it must be conceded, I think, that if we are ever to accomplish anything in this baffling field, realization can only come by intervention at the very inception of the trouble—before the obliteration of important histological elements, which, once destroyed, are never built up again.

On this point pathology and therapeutics are in agreement.

Granting even that, discouraged by the ill success of the past, we have come to look into the future in a spirit of pure nihilism, so far as the curability of this class of diseases is concerned, it were well, at least, to concede that much may be done to ameliorate and

something even to retard their course; and that certainly the latter—holding back the disease or delaying its forward movement—is best achieved before the pathological momentum, the morbid *vis à tergo*, has made irresistible headway.

But, aside from therapeutic considerations, present or expected, the element of early diagnosis is often invested with peculiar medico-legal significance. A prisoner, charged with murder, let us say, is arraigned for trial. For the lack of something better, his counsel sets up the plea of insanity. Medical experts are called in; the prisoner's mind is ransacked; and, what is proper enough in its way, he is put through what purports to be, and sometimes is, a rigid physical examination. The purpose of the last-named expedient—aside from any scientific data it may yield—is frequently largely judicial, the intention being to impress the jury, who might, perhaps, remain unconvinced by the purely psychological testimony, that the prisoner has grave organic disease of the central nervous system, which of itself proves that he must be *non compos mentis*. In assuming this position attorneys do not plant their feet nor base their hopes upon the pathology or clinical experience which are offered to the view, but upon the almost universal delusion of the lay mind that organic disease of the brain carries with it insanity as a usual, not to say inevitable, consequence. And you may be sure—especially those of you who have ever tried it—that to break down the ingeniously wrought defenses of this nature is to put your erudition, your logical acumen, and your powers of exposition to the touch of the severest proof.

Are there absolutely no signs of a single well-devel-

oped disease of brain or cord, then incipency is invoked; the prisoner is said to be at the threshold of dire morbid calamity—only one foot, 'tis true, as yet, across the sill, but presently, aye, even while the court sits, he may be expected to lift the other after! Sometimes, however—and this bears upon the trend of the present argument—the signs are perceived to be either too unsubstantial or too conflicting to permit of their being safely brought to court as testimony in support of the incipency of a single disease. Then it is that the attorney and his medical ally set up their master work—the plea of multiple incipency, the plea that the accused harbors the first stealthy beginnings not of one disease but of half a dozen!

Yes, unsatisfied with the contention that the signs point to organic implication of the nervous system of some kind and resting there, they invoke a kind of fantastic particularization, naming each disease with much show of emphasis, and parceling out the signs, say one to each disease; or, when there are not enough to go round, using the same signs as indubitable evidence of the presence of three or four different diseases, ignoring altogether the pivotal question of characteristic grouping, which, indeed, they would fain avoid, since the signs available are usually either so few in number, so conflicting, or so shadowy as to daunt even the most brazen casuist. And this is important, both from the view-point of the jurist and that of the medical witness; for it should be appreciated that temerity of this kind is best met by bringing forward the symptom groups generally admitted to be characteristic of the incipency of each alleged disease, and then revealing the full extent of the discrepancy by comparing these

symptoms, group by group, with the imperfect or specious grouping set up by opposing counsel. When such a shallow attempt to establish the presence of several diseases is not revealed in the direct testimony, it may and should be brought out by the cross-examination.

Familiarity with the signs of incipient organic disease of the brain and spinal cord is desirable, then, from a clinical standpoint, and may rise to the dignity of a positive necessity when we are called to testify in a court of law.

Unfortunately, our knowledge is not yet sufficiently advanced to admit of a codification of the signs of incipency applicable to all diseases of the nervous system. For this reason, and also to avoid the perils of a long and complicated disquisition, I shall limit the present paper to the discussion of the symptoms that proclaim with some approach to certainty the beginning of the following diseases: locomotor ataxia, multiple sclerosis, paralysis agitans, and general paresis—diseases which play a considerable part in the litigation of the day.

To win to an intelligent interpretation of the symptomatology of locomotor ataxia it is necessary to remember that the brain, spinal cord, and nerves, especially the cranial nerves, may all be implicated.

Among the possible symptoms of early tabes are paralysis of the abducens, atrophy of the optic nerve, paralysis of several of the extrinsic ocular muscles, and more or less involvement of the intrinsic ocular muscles. Upon the last-named complication hinge the various pupillary anomalies and the impairment of accommodation. Sometimes the pupils are uneven in

size; at others they are greatly contracted. Again, they may fail to react to light, but respond to efforts at accommodation (Argyll Robertson symptom). More rarely there may be total absence of response to either light or accommodation. This condition was well exemplified in a man afflicted with incipient tabes, whom I recently saw in consultation with Dr. F. Le Roy Satterlee. Tactile and thermic sensibility were but slightly if at all affected in this patient; the patellar tendon reflex was, however, lacking, and he suffered from gastric crises and occasional pains in the legs of the characteristic lancinating type. There was no apparent insufficiency of innervation of the muscles. All movements of the hands and legs were normally executed, and Romberg's sign—swaying when standing with closed eyes—was but slightly manifest.

It remains to note that the contracted pupils of tabetic subjects may often be made to expand by smartly irritating the skin with the faradaic brush.

Only exceptionally are impairment of color appreciation and diminished acuity of vision associated with a normal visual field. Again, it is worth remembering that a narrowing of the visual field is sometimes observed in the absence of all apparent neuroretinal changes. My friend Dr. David Webster has given me an account of a case of this kind observed by him—a central scotoma of the left eye, without appreciable ophthalmoscopic lesion, occurring in a man of forty, in whom, several years afterward, there developed unmistakable symptoms of ataxia.

Besides the cranial nerves just mentioned, several others may become involved in ataxia. Disturbances pointing to implication of the vagus, the accessorius, the

olfactory, the glossopharyngeal, the auditory, and the hypoglossal have all been recorded. Lesions of the facial, the trigeminus, the auditory, and the olfactory are relatively rare; while, on the contrary, implication of the vagus and the accessorius or their nucleus are to be counted among the early and common features of the disease. It is to the involvement of the last-named nerves—the vagus and the accessorius—that the gastric and laryngeal crises, the paroxysmal vomiting, coughing, and suffocative attacks are usually ascribed.

Of great interest from a clinical and medico-legal standpoint—and I can not forbear from a passing mention of them—are the cerebral manifestations and associates of tabes. Attacks of vertigo, depression, and morbid anxiety are among the lesser phenomena; delusional melancholia, dementia, paranoia, and, above all, paresis among the greater. In some instances the paresis precedes the ataxia, in others it comes after. Desirable as would be the determination of the exact sequence, this is often quite impossible, especially in medico-legal cases, because of the imperfect clinical history available.

We may well pass over the hypoglossal lesion, expressing itself in hemiatrophy of the tongue, since there is nothing in the least distinctive about it, the disease being more frequently met with as an independent affection than in association with well-marked ataxia. Indeed, much the same reservation, though somewhat more limited in degree, is applicable in the case of all the nerve lesions previously considered. Though significant, they are not in themselves determinative; only when associated with other symptoms do they attain their full diagnostic value.

What, and how many, then, are the symptoms which when associated with the phenomena induced by these nerve lesions enable us to proclaim with confidence that we are, in truth, confronted with true ataxia?

In my opinion, the presence of at least two additional symptoms is necessary to a decisive conclusion: First and foremost, the lancinating pains, with which all are familiar; and, secondly, absence of the patellar tendon reflex. These symptoms, especially when associated with immobility of the pupil, lend the ultimate of certainty to the diagnosis. Ataxia, and only ataxia, can it be, since in that disease alone are three symptoms of such striking dissimilarity to be found. So much for tabes.

The next disease, multiple sclerosis, or disseminated sclerosis, to which I would briefly advert, is not only a rather rare affection of the nervous system, but also one characterized by an obscure and insidious beginning. Moreover, in its subsequent evolution, it is frequently baffling and inconstant. Seldom do we meet with absolutely typical symptoms. The very early symptoms—vertigo, gastric derangements, headache, and minor sensory and motor disturbances—are, certainly, in no sense distinctive. We may, of course, suspect that a nervous disease of some kind is developing; but only with the appearance of the intention tremor—that form of tremor brought on alone by purposive acts or attempts at them—do we begin to fully realize the nature and extreme gravity of what lies before. The subsequent appearance of nystagmus, which represents merely an extension of the intention tremor, and the scanning speech, characterized by a drawling hesitancy between

words and letters, complete what, for the books, is the typical symptom-group of the disease.

Aside from occasional paræsthesias, sensory disturbances are absent in multiple sclerosis.

On the other hand, muscular rigidity, increased skin and tendon reflexes, and a spastic gait are frequently met with. This spastic condition was strikingly in evidence in a young girl of sixteen, whom I saw in consultation with Dr. Stephen Pierson, of Morristown, New Jersey, a few years since. Tendon reflex and ankle clonus were as exaggerated as possible; the characteristic speech defect was present; and there was besides decided evidence of commencing dementia. But, on the other hand, of nystagmus there was practically none at all.

One or more of the cranial nerves is also frequently implicated. This fact has not the significance it possesses in tabes. The differential diagnosis is, indeed, quite simple when the course of the disease is in other respects fairly typical. Again, slight dementia and apoplectiform attacks are sometimes observed, the latter more especially toward the beginning of the disease, while the extension of the disease to the cortex has obviously to do with the genesis of the epileptiform seizures.

Where the diagnosis is beset with difficulty, as it often is in this disease, it would be but a betrayal of pedantry to attempt too much in the way of early augury. Enough, then, that the appearance of the intention tremor justifies the gravest suspicions—suspicions which deepen into certainty with the coming of nystagmus or marked exaggeration of the tendon reflexes.

While locomotor ataxia is a disease of the general nervous system with a predilection for the spinal cord, paresis, on the contrary, ravages by preference the brain, attacking the spinal cord and nerves much less sharply. Since in the clinic and in court the question often arises as to whether in a given case we have to do with early paresis or not, you will agree with me that a few minutes given to the recapitulation of the more important data will put a fitting ending to the discussion. In the beginning, as in the later stages of the disease, psychical changes play a prominent part. The subject is lethargic in thought and perception; he has likewise parted with his former power of attention, and is therefore unable to persist in his work for long. He is irritable and fretful; his equanimity is jostled by trivial circumstances; his will is weak, and he is lightly swayed in his fickle determinations. Still more significant is his neglect of the reasonable conventions and even the decencies of life. Formerly a respecter of cleanliness and decorum, he is now become slovenly in his raiment and indecent and obscene of speech. As a rule, he is quite unaware of this change of character; but exceptionally—and this applies especially to medical men, who are aware of the insidious nature of the disease—he may strongly suspect that all is not well with him, and communicate his suspicions to his friends.

All this is extremely significant in a general way; but it is not quite conclusive, for in mania and other forms of mental derangement there may be in the beginning striking alterations of appetite, habit, and propensity. Nor yet is the nocturnal insomnia nor somnolence by day decisive. Presently, however, or exceptionally only after the lapse of years, certain other

symptoms of a somatic character are added. Prominent among them are alteration of speech. Words are brought out with difficulty; syllables and letters are misplaced, and there is a distinct loss of vocal timbre. This is the "syllable stumbling" of the German writers. To these derangements are soon added fibrillary trembling of the lips and tongue, inequality of the pupils, changes in the writing—manifested by irregularity and dropping of letters and syllables—and clumsiness of gait.

Clinically speaking, all the elements necessary to a diagnosis are now present, and the case may be declared without further hesitation to be one of incipient paresis. In medico-legal cases, however, matters are not always so simple. The inequality of the pupils may be produced artificially; the subject may be taught to shuffle about and affect the necessary idiosyncrasies of speech and conduct; and twitchings of the muscles may be produced by the aid of strychnine. All this opens a vista of unique possibilities.

The subsequent course of a case of paresis is known to all: how the subject, parting with his critical faculty, gives himself over to the dominance of the most quixotic schemes and extravagant, unsystematized delusion; how the pupils become absolutely inactive, the sensibility of the skin diminished, the tendon reflexes lost, the optic nerve atrophied, and the cranial nerves, or some of them, paralyzed.

Some or all of these things may happen, as all know, from reading and experience. Finally, the epileptiform and apoplectiform attacks, with their false recoveries, the bedsores, the relaxation of the sphincters, and the terminal dementia are things well known to those who have had asylum experience.

A word more on the principles of differential diagnosis applicable in the clinic and in court and I have done. Paresis, as everybody knows, does not always follow the classical course just indicated. Sometimes the symptoms point to progressive mental deterioration from the very beginning, dementia being an initial rather than a terminal feature. Again, the disease in its course may bear more or less resemblance to a number of other affections, notably chronic alcoholism, cerebrospinal syphilis, senile dementia, chronic meningitis, multiple sclerosis, and exceptionally even tumor of the brain has been mistaken for it.

In alcoholism, the disturbances of speech are less characteristic, the hallucinations assume a unique and prominent character, and the tremor is increased by abstinence and decreased under the influence of alcohol. Then, too, the history of the case is of service in forming a decision.

A sharp distinction from cerebral syphilis is often impossible; indeed, syphilis is generally conceded to be, as in ataxia, one of the chief causes, if not the chief cause, of the disease, and this quite aside from any hereditary predisposition. The characteristic splitting, persistent headache of cerebral syphilis is, of course, of some diagnostic service. In senile dementia the absence of important somatic criteria and the age of the patient are usually sufficient for purposes of exclusion. The comparative frequency of choked discs and the occurrence of delirium and fever in the beginning will help us to exclude meningitis. In multiple sclerosis the intention tremor, the nystagmus, and the scanning speech of fully developed cases make error impossible. When, however, the development of the disease is

erratic, distinction is sometimes impossible. The absence of mental symptoms and pupillary changes, the attitude, the deliberate speech, quite unlike the precipitate scanning utterance of paresis, will serve to distinguish paralysis agitans.

What adds further complications to the problem of diagnosis is the fact that paresis frequently coexists with other diseases. This fact, as already noted, has been utilized by counsel in cases involving medico-legal questions with a view to mystifying the jury, the ultimate purpose being to imbue their minds with the idea that even though the attempt to prove the existence of paresis falls through, enough evidence still remains to establish the existence of grave organic disease of the cerebro-spinal system. And, as previously intimated, there being a popular idea that organic disease of the nervous system necessarily entails insanity, it requires little in the way of artful presentation to win to a favorable verdict, borne thither on a wave of maudlin eloquence, whose meretricious sheen casts in obscurity the poor protest of simple fact.

When a contention of this kind is set up, its speciousness is best revealed by insisting that the minimum of symptoms possible to the constitution of the typical group of each disease be produced in evidence. For example, when, as often happens, paresis and ataxia are alleged to be associated, the characteristic cerebral symptoms of the former should be demonstrable, and the spinal symptoms of the latter. True, both are diseases of the general nervous system; but, whereas in paresis the brain is chiefly attacked, in tabes it is the cord which is predominantly involved. This proposition being admitted—and pathologically it is incontrover-

tible—it follows that the decisive symptoms in paresis are cerebral, in ataxia spinal.

Many a web of fine-spun sophistry will vanish into smoke, you may be sure, at the touch of such simple logic.

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